SHORT REPORTS

Chronic paroxysmal hemicrania: first reported British case

We report the first British case of chronic paroxysmal hemicrania, a rare but debilitating form of headache in which treatment with indomethacin immediately abolishes the attacks.

Case report

A 22 year old woman was referred with an 11-year history of headaches. These had occurred every day apart from during two periods, one when she was 13 and one when she was 16, when she had been free of headaches for three and six months respectively. During the first five years the headaches had lasted for only 15 minutes and had rarely occurred more than once a day, but they had progressively become longer and more frequent. At presentation they were lasting one to two hours each day, during which time she had several attacks of excruciating pain that lasted for five to 10 minutes, with a duller pain between each exacerbation. She therefore had 15-20 attacks each day, a large proportion of which occurred at night.

The pattern of the headache was constant. It began as a severe sharp stabbing pain above the left ear and in a few seconds spread to the whole of the left side of the face, left anterior scalp, and occasionally around the left common carotid artery. It occurred at any time and woke her from sleep three or four times each week. It was associated with lacrimation of the left eye, blockage of the left nostril, and left ptosis. It could be precipitated by movements of the neck.

There were no abnormal signs on examination apart from bruising of both upper arms where she gripped herself during attacks. When observed during an attack, precipitated by neck flexion, she developed appreciable bradycardia (40 beats/minute, regular) despite being in severe pain. Left ptosis and oedema of the upper eyelid and ipsilateral pupillary constriction developed within two minutes of the start of this pain.

She was fully investigated on several occasions. A computed tomogram, skull and sinus *x*-ray films, and an electroencephalogram were all normal. No dental abnormality had been found, although impacted wisdom teeth had been extracted in 1978 with no change in the frequency or severity of the headaches, which did not respond to any analgesics. She had been treated with many drugs without success.

Chronic paroxysmal hemicrania was diagnosed and indomethacin 25 mg thrice daily prescribed. The headaches immediately became much milder and stopped totally after five days. They returned briefly three months later during an episode of severe gastroenteritis,[1] when presumably the indomethacin was not being absorbed, but were relieved as soon as she recovered. She remained symptom free at follow-up after nine months.

Comment

Chronic paroxysmal hemicrania was first described in a preliminary report in 1974,[1] with a full report in 1976.[2] A review of the condition in 1980[3] included eight well-documented cases and 10 possible cases. There are thought to have been about 40 cases world wide (O Sjaastad, personal communication).

Chronic paroxysmal hemicrania may be differentiated from ordinary cluster headache by three features. It occurs predominantly in female patients; many attacks occur each day (15 or more in 24 hours), which are of shorter duration than cluster attacks; and there is a prompt and complete response to indomethacin.

As in our patient there is often a phase during which the pattern of attacks is atypical. Indomethacin is the only non-steroidal anti-inflammatory drug that has been shown to be effective in this condition; it is not effective in ordinary cluster headache though is useful in other forms of headache—for example, cluster headache variant,[4] in which numerous brief, knifelike pains occur in several parts of the head and there is prolonged benign exertional headache.[5] The required dosage of indomethacin varies considerably, most patients responding to 75 mg a day, although occasionally 250 mg a day or as little as 12·5 mg a day are necessary; thus it is important to titrate the patient's response against the dosage.

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Dihydrocodeine narcosis in renal failure

Dihydrocodeine tartrate is commonly used to relieve mild to moderate pain. We report a case of severe narcosis caused by the drug in an anuric patient receiving maintenance haemodialysis.

Case report

A 41 year old woman was admitted for ligation of unsightly veins around her Cimino fistula after six years' maintenance haemodialysis. Preoperatively she received pethidine 50 mg and promethazine 25 mg, and postoperatively two doses of papaveretum (15 mg and 10 mg). The anaesthetic was halothane and nitrous oxide. She made a good recovery and over the next four days received dihydrocodeine 60 mg by mouth three times daily (total 600 mg) to relieve pain.

Her Cimino fistula became unusable, and on the fourth postoperative day peritoneal dialysis was begun via a catheter inserted under local anaesthesia. No premedication was given. During the following 24 hours her condition deteriorated. She developed myoclonic twitching and became unrousable and hypotensive. Her pupils were constricted, suggesting opiate toxicity. Intravenous naloxone 0·4 mg produced a dramatic improvement: her pupils dilated, she woke and hyperventilated, and her blood pressure rose. The improvement was not maintained, and she required multiple injections of naloxone and eventually a continuous infusion of 0·4 mg/hour, which maintained respiratory and circulatory function (total 8·0 mg in 48 hours).

The following day she became acutely hypertensive (blood pressure 200/140 mm Hg) and developed grand mal convulsions. A computed tomogram was normal, as was her cerebrospinal fluid. Naloxone was stopped and her blood pressure returned to normal, but she required ventilation.

Three days after the last dose of dihydrocodeine she was still suffering from narcosis; her plasma dihydrocodeine concentration was 0·7 mg/l. Extubation became possible on the eighth day, when her plasma dihydrocodeine concentration was 0·04 mg/l. Her liver biochemistry remained normal throughout.

Comment

Dihydrocodeine was first prepared in 1911, yet little is known about its pharmacokinetics and pharmacodynamics.[1] Its unwanted effects—namely, respiratory and circulatory depression, nausea, vomiting, and constipation—are similar to those of morphine but less pronounced.[2] Although our patient had received other centrally acting depressant drugs four days earlier, the temporal relation of events suggested that her clinical state was due to dihydrocodeine toxicity. The drug concentration was measured by gas chromatography, which discriminates between dihydrocodeine, naloxone, and dihydrocodeine metabolites.

A dihydrocodeine concentration of 1 mg/l has been associated with narcosis, and the concentration of 0·7 mg/l at 72 hours suggests that at the onset of narcosis accumulation of the drug had led to toxic concentrations. Other mechanisms may, however, be postulated, as signs of narcosis persisted and she remained responsive to naloxone despite lower drug concentrations. Such mechanisms include displacement of protein-bound drug in uraemia, resulting in higher free concentrations;

the accumulation of active metabolites; and an interaction between dihydrocodeine and endogenous opiate-like peptides. Levine[3] described an increased narcotic effect of codeine phosphate associated with hypocalcaemia in renal failure; our patient, however, remained normocalcaemic. The convulsions might possibly have been due to toxic dihydrocodeine concentrations,[4] but hypertensive encephalopathy was a more likely explanation in the present case.

In conclusion we urge caution in prescribing dihydrocodeine in conventional dosage to patients with severely impaired renal function.

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Perinatal transmission of hepatitis B in Northern Ireland

Hepatitis B infection may be transmitted from the mother to her newborn infant, so that the infant becomes a carrier. The transmission rate varies from 0 to 6% in Europe and North America[1] to 40% in Taiwan.[2] e Antigen (HBeAg) is associated with a high risk of transmission,[2-4] and the high prevalence of HBeAg positive carriers among Chinese and Asian racial groups may account for this difference. The Northern Ireland Blood Transfusion Service routinely tests roughly 98% of antenatal samples from the region, and screening for hepatitis B surface antigen (HBsAg) has been included since 1973.

Subjects, methods, and results

We undertook a retrospective study of women positive for HBsAg and their children using radioimmunoassay (Blood Products Laboratory). The number of births during 1973-81 was 244 887 in a total population of 1 541 987, the immigrant population forming only a small proportion. All sera positive for HBsAg were referred to the Regional Virus Laboratory for confirmation and, from 1981, to the West of Scotland Blood Transfusion Service for marker studies (radioimmunoassay; Abbott) of antibody to hepatitis B core (anti-HBc) and HBe antigens. Cord blood tests were not possible until 1981, and a child was considered to be positive only if markers persisted after the age of 9 months. The study included all older children in the family when an antenatal sample was HBsAg positive.

Fifty two HBsAg positive mothers were identified: 34 were white, 16 Oriental, and two African. The table gives the results in children born to 17 of the 34 white mothers. One and possibly two of the white mothers positive for HBeAg had transmitted infection to her child. There was also evidence of transmission in case 3; the woman developed anti-e one year after delivery. HBe studies were not done in case 4 until after the birth of all the children, and the woman may possibly have been an HBeAg carrier during her pregnancies.

Carrier state of families of white mothers positive for HBsAg (children positive for HBsAg are described by their HBe state alone. The two children described as having anti-HBc had no other hepatitis B markers)

Case No	eAg	Anti-e	Transmission
1	+	−	1 child HBeAg positive
2	+	−	1 child anti-HBc only at 9 months
3	−	−	1 child HBeAg positive
4	−	+	2 children HBeAg positive 1 child anti-HBc positive at 3 years
5	−	+	1 child immune 2 children no markers
6-17	−	+	No markers among 16 children

Comment

Data from other parts of the UK suggest that, apart from infants of women with acute hepatitis B during the last trimester, newborn white infants are not at risk. All the women studied here had been carriers for several years, and there was no history of acute hepatitis during pregnancy. Our experience indicates that, at least in Northern Ireland, an appreciable number of white children have been infected by their mothers (seven of 25 children studied showed hepatitis B markers).

Reliable tests for HBe markers have become available only recently; we do not, therefore, have records of HBe state at the time of the relevant pregnancies in cases 4 and 5. One of the women positive for anti-HBe had a sister who was also an HBsAg carrier with anti-HBe. Each had two unaffected children, but the mother of the proposita was HBsAg and anti-HBe positive. Possibly the mother may have been a carrier of HBeAg in the past.

Our findings indicate that there is a substantial "high risk" group of white people in some communities, and any discussion of prophylaxis for neonates should not, therefore, exclude them. The presence of e antigen and the absence of all e markers continue to be indicators of a high risk of transmission. All babies born to mothers in either of these categories should be offered prophylaxis. We have initiated a protocol of passive-active immunisation for high risk babies in Northern Ireland and hope to collaborate with regions in Scotland. The protocol includes whites and non-whites.

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Gangrenous caecal volvulus after colonoscopy

Although potentially hazardous, colonoscopy is generally safe with few complications.[1] Perforation and bleeding are the most common complications, but intramural air dissection, retroperitoneal emphysema, splenic injury, asymptomatic serosal tears and mesenteric haematomas, ileus, partial volvulus of the transverse colon, and incarceration of a hernia have also been reported.[1-3] We report a case of caecal volvulus after colonoscopy.

Case report

A 71 year old woman was admitted with a history of weight loss, malaise, bleeding per rectum, and intermittent fever. On examination she was feverish and the colon in the left iliac fossa was tender and palpable. Tests for faecal occult blood were positive on three occasions. Barium enema showed numerous diverticula, particularly in the descending and upper sigmoid colon. Barium was noted outwith the colon at the level of the iliac crest, and in the same area there was a suspicion of an intraluminal mass lesion. She was referred for colonoscopy.

After 48 hours of full routine preparation colonoscopy was carried out with an Olympus TCF type 2L2. Fluid faecal residue in the lower colon necessitated frequent aspiration and slow advancement of the instrument, but the splenic flexure was reached without problem. No intraluminal lesion was seen up to this level. Over the next 48 hours she complained of increasingly severe central abdominal colic and abdominal distension developed. She was transferred to the surgical unit. Abdominal examination showed